# VALIDATION OF THE SHARED DECISION-MAKING MODEL IN THE CONTEXT OF A PATIENT PRESENTING TO THE EMERGENCY DEPARTMENT WITH CHEST PAIN OF POSSIBLE CARDIAC ORIGIN

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By D. F. Flaws

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# Abstract

The intention of this thesis was to investigate the feasibility of clinical shared decision-making. If physicians are to contribute to a shared decision process, they will need to be able to communicate unbiased information to their patients clearly. Thus, physicians need to provide some form of quantitative risk estimate.

Physicians estimated the probability that a patient presenting with chest pain had Acute Coronary Syndrome. The patients details were then entered into a structured clinical risk calculator and the results were compared.

It was found that although both methods held comparable predictive power, the physician's estimate did not correlate well with the structured estimate. This suggests that physicians do not utilise a quantitative risk estimate. It then found that the correlation between risk estimates increased as further investigations were performed. However, neither estimation method could predict these test results.

It was hypothesised that physicians utilise a dichotomous decision process. Thus, as positive and negative test results erode the intermediate risk group and populate the high and low risk groups, the correlation between methods increases.

It was concluded that a dichotomous decision process would provide a considerable hurdle to the shared decision-making process, as it would limit the communicability of the physicians thought process. However, the potential benefits of shared decision-making encourage future researchers to find a way to overcome this hurdle.

# Introduction

The intention of this study is to investigate whether physicians are capable of sharing the diagnostic decision process with their patients by comparing the decision process of an Emergency Physician in deciding whether to admit or discharge a patient having been investigated for Acute Coronary Syndrome (ACS) against a structured risk estimate (in the form of a risk calculation program), and to measure physician reliability in providing numeric probability estimates based on risk factors.

## Decisions in Medicine

In general, medicine has two situations where a decision will be made: The decision of how to test for a condition, and the decision of how best to treat a condition (Pauker et. al., 1980). It is important to realize that in both of these situations, the option to withhold further testing and treatment is also considered. Effectively, the decision process is the same in each case: Do the benefits of any given course of action justify the potential risks and costs involved? In cases where multiple treatment options are available (including the option to withhold treatment), the management pathway with the best cost/benefit ratio is considered the best alternative.

With this in mind, a body of literature has grown from the 1980's onwards around deriving a "testing threshold equation" (Pauker et. al., 1980). By calculating these cost/benefit ratios (based on patient symptoms, and large databases of patient outcomes), it is possible to assess whether any given test or treatment plan is in the patient's best interests. For diagnostic scenarios a "testing threshold" can be identified.

The testing threshold is generally taken to be the point where the risks from further investigation are equal to the risks of ceasing investigation (Pauker et. al., 1980). Thus, any presentation for which the pre-test probability falls below the testing threshold is not further investigated (as the risks of investigation outweigh the risks of the disease), and any presentation for which the pre-test probability falls above the testing threshold is investigated further (as the risks of a missed diagnosis outweigh the risks of the investigations).

This theory has been highly influential, and has led to the development of several medical "decision aids" (Kline et. al. 2005 & Wells et. al. 2000). These aids are generally derived by measuring various risk factors in a large observational study. Subsequently, an analytical process called recursive partitioning results in the identification and weighting of the independently influential risk factors. These are used then to design a simple rule to stratify patients into risk groups, or suggest how to best manage any given patient (Wells et. al., 2000).

Decision aids that have developed from the testing threshold theory have an important place in modern medicine because they combat the well-established fact that unaided doctors are subject to a myriad of biases and heuristics (Gigerenzer et. al., 1995 & Sox, 1986). However this approach to medical decision-making is based on the critical assumption that all patients value all risks, costs and benefits equally. A blanket approach where physicians make decisions on behalf of a patient (but without their direct input) is often referred to "paternalistic" (Back et. al., 2006).

**Decisions in Other Fields**

In the non-medical fields of psychological research, particularly in the industrial sector, a large body of literature on decision analysis and the perception of risk has been developing since the 1950's. It is now well understood that individuals vary considerably in their risk behaviour (Mayo et. al., 1990). For example, if a group of people planning a family holiday were asked if they would consider flying, a number would be reluctant, possibly due to the potential of a plane crash; and yet many of the people who refuse to travel by air would be prepared to drive a car on a highway, even though it is the statistically riskier activity (Mayo et. al., 1990) Two important observations can be made from this example.

The first observation is that a decision that at first glance may appear to be irrational or even hypocritical judgement is in fact a highly logical decision in what is considered an acceptable level of risk. The risks involved in operating a motor vehicle, though statistically more salient, are largely under control of the stakeholder; the stakeholder has control of their speed, which highway they take, and when they stop. In contrast, the risks involved in flying are largely beyond the stake-holders control and the potential negative outcomes more catastrophic (Mayo et. al. 1990).

The second important observation is that other individuals will be prepared to fly, fully understanding the risks involved, and yet refuse to drive. People's personal values will influence what risks and benefits they consider important, and thus in which activities they are prepared to participate. For example, some people who picket against smoking, may choose to sunbathe, while other people who choose not to sunbathe may happily smoke a cigarette. It is important to recognise that the difference in risk behaviour is not necessarily caused by one party acting irrationally, or failing to understand the risks, but by a fundamental difference in personal values.

These two observations have significant implications for medical decision-making. To provide an applied example, a certain treatment could leave the patient unable to work for a month (the testing threshold theory does not consider this outcome as significant). Some patients would consider the loss of potential earnings irrelevant, and not factor that into their decision (as assumed by the testing threshold theory); while other patients will consider the economic impact carefully before making a decision. Some patients may be prepared to accept a higher risk of death in order to avoid the discomfort involved in loss of earnings.

**Implications for Medical Decision-Making**

Differences in individual values are not accounted for very well by testing threshold theory. In terms of calculating risk, decision aids are not able to weight different risks and benefits according to the unique values of specific patients. In general, the principal shortcoming of more paternalistic approaches to medical decision-making is that the entire patient populace is treated with a single value-set (or the values of their physician), and variation between different people's values remains under-accommodated.

Another finding from human decision-making research is that people are generally prepared to accept a higher level of risk when they are not the stake-holder in the decision (Mayo et. al. 1990). This is of particular relevance to medicine, as the medical practitioners are making decisions that will impact more on their patients than on themselves.

When a group of doctors at a New Zealand Emergency Department were asked what potential consequences they consider important when making a medical decision, many doctors considered the possibility of a malpractice claim (the risks for their own welfare) as

of equal importance as the patient's outcome (Flaws et. al. 2008). Understandably this value was not reciprocated in the patients (the stake-holders) that were surveyed, who considered the doctor's welfare from the decision of little importance in comparison to patient outcome.

In fields such as industry and agriculture, it is often appreciated that both parties hold a piece of the decision-making puzzle, and contemporary risk based decisions (such as whether or not to build a chemical plant near a playground) are often made from the combined input of experts (with all of the facts), and the local stake-holders (with all of the values) (Mayo et. al. 1990). However, many medical practitioners still subscribe to the paternalistic approach.

Unlike the types of decisions upon which conventional decision theory has mostly been based, the physician is making a decision on behalf of a third party, and thus in this scenario, the decision-maker is not the primary stakeholder (Davis et. al. 1996). Thus, where most decision-makers are able to simply evaluate potential outcomes based on their own values, the physician must be able to evaluate the potential outcomes of the decision based on the values of the third party. Traditionally, it has been assumed that physicians are competent at performing this decision task; that they will accurately interpret and process the risk data in front of them, and consistently elect the options which are in the patient's best interest (Epstein et. al. 2004 & Finucane et. al. 2005). However, some now argue that this paternalistic approach should be revised and that any clinical decision involving risk for a patient should be made with input from the patient; that it cannot be assumed that the physician is capable of discerning the patient's best interests, as this is solely based on the individual patient's values (Hibbard et. al. 2005).

**Shared Decision-Making: A Novel Decision Model**

Thus, the concept known as “shared decision-making” has arisen to challenge paternalistic models of medical decision-making, such as the testing threshold. Shared decision-making divides the decision process between the physician and the patient. The physician's role in the decision is to use their medical expertise to provide and communicate reliable information to the patient on all the possible options, and the risks for each option. The patient's role is to then make a decision based on this information, and their own personal values (Calman et. al 2002).

In order for this process to work, a number of assumptions must first be validated. The first assumption is that physician values are significantly different from patient values, and that the physician is incapable of incorporating the patient's values into his or her decision. This would indicate that the decision process can benefit from patient input. The second assumption is that physicians are capable of communicating all of the necessary information to their patients in an effective enough manner for the patient to make an informed decision. The third assumption is that the patient will then be capable making an informed decision, and communicating their response back to their physician effectively. Overall, this proposition is problematic, because as much as disregarding patient values may be considered unethical, it has also been found that a truly informed patient opinion can be very difficult to obtain (Bowling et. al. 2001 & Hibbard et. al. 2005).

The medico-legal implications of miscommunication must be considered. In a situation where a patient elects a management strategy traditionally considered suboptimal (involves a higher risk of death, or morbidity than alternative strategies available), and subsequently suffers death or disability, there are serious implications. If the patient elected the traditionally sub-optimal strategy as a truly informed stakeholder, then the negative outcomes

were simply risks they had accepted. This should be treated in a similar manner to a patient refusing to attend their General Practitioner, or choosing to smoke.

However, if it can be indicated that the patient was not adequately informed when making the decision, then the responsibility for negative outcomes would lie with their physician. Perhaps the greatest problem with this situation is that it is no different to the paternalistic approach. Thus, if communication is insufficient, the shared decision-making model reverts back to a paternalistic model.

Given the serious implications of miscommunication in a shared decision-making model, physicians would deserve some assurance that any protocols implemented provide them some form of security in the case of negative outcomes. Otherwise, the physician is as liable for adverse outcomes as in the paternalistic model, but without the same control. Thus, it is possible that if the physician has any doubt as to whether their patient has truly understood the implications of their decision, the physician may naturally revert to a paternalistic approach as a means of self protection.

For this reason, the primary intention of this thesis is to investigate the second assumption; that physicians are capable of communicating risk information to the patient in a format that is comprehensible. If the physician cannot explain their risk assessment verbally, or they cannot effectively express the probabilities they have used in their own decision process, then it is unlikely (perhaps impossible) that they will be able to communicate the information necessary for the patient to perform their own values-based assessment.

**Using ACS as an Example of Medical Decision-Making**

Acute Coronary Syndrome (ACS) is a good example of a condition that raises many of the issues illustrated above. ACS describes a clinical scenario in which the Coronary Arteries (which supply the heart muscle with oxygen) become gradually become narrowed to an extent that can cause insufficient oxygen supply to the heart muscle. Depending on the extent of the oxygen insufficiency a spectrum of severity can occur from chest pain (angina) through to heart muscle death (a heart attack). ACS typically presents with chest pain but this is a common symptom in many conditions (Green et. al. 2000).

ACS is a difficult condition to manage, as its prognosis is serious and its signs and symptoms are difficult to differentiate from less harmful conditions (Hollander et. al. 2004). To further complicate patient management, the preliminary tests have a poor diagnostic accuracy, and the later, more specific and sensitive tests have higher risks of causing harm (Cowan et. al. (1994) & Devlin et. al. 2005). Thus, when a patient presents to the emergency department with potential Acute Coronary Syndrome, the decision of whether to continue investigation is necessarily a risk-based assessment (Hollander et. al. 2004). Patients present to Christchurch Hospital with symptoms suggesting ACS on a regular basis. Previous local investigations have shown that, of the 4,000 patients that are investigated for ACS in Christchurch Hospital each year, approximately 23/% are diagnosed with ACS, while the remaining 75% have other conditions (most of which are less serious) (local audit data, Christchurch Public Hospital).

This provides an excellent context in which to investigate some of the issues raised about a shared diagnostic decision process, as it provides a risk-based decision, with important potential outcomes, and a large population sample.

**A Derived Testing Threshold for ACS**

A recent study at Christchurch hospital assessed the levels of risk acceptability for discharging a patient with chest pain of possible cardiac origin for people with and without medical expertise, in the interest of evaluating the assertion that patient values will differ from physician values (Flaws et. al. 2008). Unfortunately, although physicians completed the surveys easily, non-experts failed to complete the surveys provided, and no statistically reliable data was obtained on this group. Communicating this information to patients is clearly difficult. In light of this, it is important to refer to the assumptions of shared decision-making mentioned earlier; particularly the assumptions that physicians can communicate the relevant information effectively, and that the patients can effectively perform and communicate back their decision. This preliminary result already draws these two assumptions perilously into question. It was tentatively concluded that, if a patient is unable to make a numerical expression of acceptable risk, then practitioners may have little choice but to base their decisions on medical opinion. In this particular instance, physicians felt a a risk level of 2% or above was considered unacceptable to discharge a patient who presented with potential Acute Coronary Syndrome. A calculated value, based on Pauker & Kassirer's equation (1980), also found the ACS testing threshold for Christchurch Hospital to be 2%.

Although this study found a statistically reliable value for physician opinion, this opinion has never been compared to physician behaviour. It may be, for example, that although physicians state that 2% is an unacceptable risk for discharge, that in practice physicians continue discharging beyond this risk level. Thus, the present study aims to compare the risk acceptability value obtained in this previous study with the value observed in physicians' actual behaviour. This study also aims to measure the reliability of the physician's estimates by comparing physician opinion to a structured risk estimate calculated on an internationally

validated risk stratification tool, in order to observe whether physicians will be able to communicate this information to their patient in a shared decision-making model.

# Methodology

*Summary*

1. Patients investigated for ACS were recruited to this study in collaboration with another study being performed at Christchurch Hospital.
2. The results of the investigations performed, as well as a 45-day follow-up, were used to blindly and independently diagnose which patients had ACS.
3. The physician who saw the patient in the Emergency Department (ED) estimated the risk that the patient was suffering from ACS before ordering an initial blood test.
4. The patient's signs and symptoms were entered into a PREtest Consult risk calculator.
5. The physician's risk estimate was compared to a PREtest Consult calculator.
6. Both estimates were validated against the patient's final diagnosis (obtained after all test results have been obtained, any relevant interventions performed, and a 45 day (post-discharge) follow-up period has been completed).

**Standard Management of Patients Suspected of ACS**

To preserve patient safety, all patients were given standard management, as described below:

*Investigations*

**In the Emergency Department (ED)**

All patients suspected of ACS in ED receive a Troponin I blood test, and an ECG

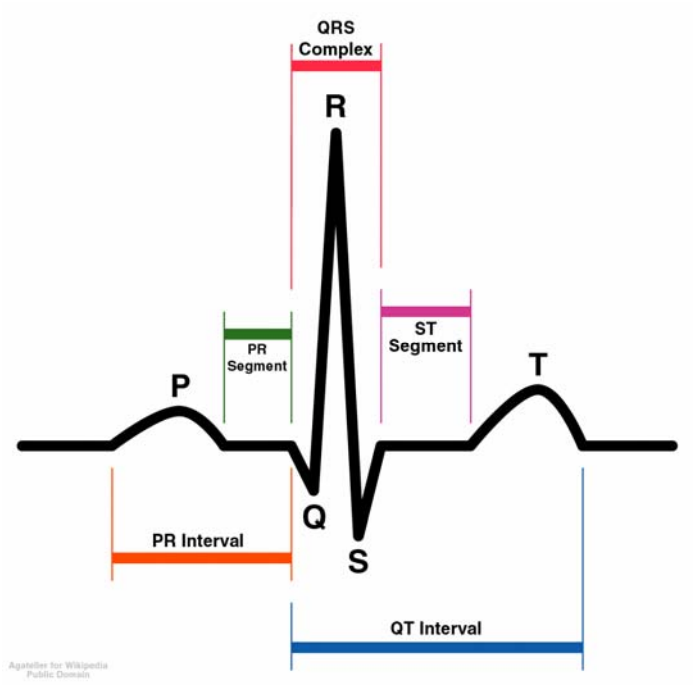
The **Troponin I** (TnI) blood test detects Cardiac TnI within the blood stream. TnI is a structural protein found within the heart muscle, which leaks into the blood when heart muscle has died. A level above 0.03ng/L is considered an abnormal result. The test is highly

sensitive to cardiac injury, but does not discriminate well between ACS, and other forms of heart disease, such as pericarditis. In fact, even physical trauma, or excessive exercise can cause troponin levels to rise.

ACS causes a characteristic rise in troponin levels over time, which distinguishes it from other conditions. Thus, serial TnI tests are performed throughout a patient's admission. All patients suspected of ACS will receive at least one Troponin I blood test upon arrival, and a second one more 6 or more hours later to see if the Troponin levels are rising or stable http://www.patient.co.uk/showdoc/23068792/ (Patient information resource on ACS management accessed 12/1/2009).

A 12-lead **ECG** traces electrical impulses from 12 different regions of the heart. The normal heart reading is divided into wave sections, known as P wave (atrial contraction), QRS complex (ventricular contraction, Q wave not evident in every lead), T wave (repolarisation), and U wave (not always evident). Although the exact waveform differs from lead to lead, Figure 1 indicates the overall pattern of a single atrio-ventricular contraction.

**Figure 1. A standard Atrio-ventricular waveform**




Systematic deviations from the standard output pattern can indicate various cardiac abnormalities. Various segments may be too far apart (long interval), too close together (short interval), sitting above the expected level (elevation), below the expected level (depression), completely upside down (inversion), or absent altogether. Various conditions result in systematic changes, allowing these abnormalities to be used diagnostically. For example, hypocalcaemia (low levels of calcium in the blood) results in a long QT interval, while hypercalcaemia (high levels of calcium in the blood) results in the reciprocal change (a short QT) http://www.patient.co.uk/showdoc/27000320/ (Patient information resource on ECG procedures accessed 12/1/2009).

Changes indicating potential ACS are deviation of the ST segment (ST-Elevation, ST-Depression), inversion of the T wave (T-Wave inversion), or Q wave abnormality (presence of a Q wave in a lead where it is not normally found). An ST-Elevation (the most serious of

these changes) was considered an exclusion criterion for this study, as these patients are always admitted as part of standard management; thus, a risk-based decision process of whether or not to discharge never took place with this elevation (Green et. al. 2000).

**After Admission**

Admitted patients can receive any combination of an Exercise Tolerance Test, Dobutamine Stress Echo, Coronary Angiogram, or Coronary Arterial Bypass Graft or further ECGs while they are in hospital, or within 45 days of their discharge. Each of these investigations carries some risk of causing undesirable outcomes for the patient, such as cancer (from radiation exposure), stroke, or even a heart attack. A number of the tests can also be quite uncomfortable.

An **Exercise Tolerance Test** is a test used to help rule out ACS in low-risk patients. The patient walks on a treadmill for as long as possible, while the speed and difficulty gradually increases. Eventually the speed can increase to the point of challenging athletes, but many patients do not progress beyond the walking stage. The patient's heart is monitored via an ECG. A sufficient test, with no ischaemic signs on ECG is considered a negative result, and helps rule out the possibility of ACS, whereas a completed test with ischaemic signs (such as ST Depression >2mm) is considered a positive result, and indicates further investigation must be undertaken. An incomplete test, or non-specific signs (such as ST depression <2mm) is considered an equivocal result, and is typically followed up with further investigation (often leading to a Coronary Angiogram or a Dobutamine Stress Echo) http://www.patient.co.uk/showdoc/27000323/ (Patient information resource for exercise tolerance testing, accessed 12/1/2009)).

The **Dobutamine Stress Echo** Test is performed on patients who are incapable of conducting an Exercise Tolerance Test. The patient's heart rate is increased to an excited level via intravenous injection of a drug called Dobutamine. The heart is monitored for ischaemic signs (such as regional wall motion abnormality) via echocardiogram. Regional wall motion abnormality is a section of the heart muscle moving in an unusual manner, and suggests that heart muscle has been damaged. The results are interpreted as positive, negative or equivocal in the same manner as the Exercise Tolerance Test, and treated accordingly http://www.patient.co.uk/showdoc/40000527/ (Patient information resource for cardiac stress imaging procedures, accessed 12/1/2009)).

Unlike the Dobutamine Stress Echo and Exercise Tolerance Tests, **Coronary Angiography** is performed on patients highly suspected as having ACS, and is the first choice of the more invasive procedures that can be performed. A catheter is inserted into the patients' femoral artery via their groin. The physician then slides the catheter through the patient's arterial system, and into the coronary arteries. A mildly radioactive "contrast" is then injected into the coronary arteries, and is detected on a real-time chest X-ray. Any artery through which the contrast passes through slowly or not at all is clearly visible, and this indicates some level of artery narrowing, or occlusion. If any artery is seen to be more than 50% occluded, the result is interpreted as positive. Otherwise the interpretation is negative. The physician then has the option of either performing a balloon angioplasty, or inserting a stent (both described later) before withdrawing the catheter. The entire procedure is minimally invasive, but does present a minor risk of inducing a myocardial infarction, or causing a stroke. There is often some heavy bleeding from the groin after removal of the catheter, which requires pressure to be applied to the point of entry for some time http://www.patient.co.uk/showdoc/27000304/ (Patient information resource for Coronary Angiography procedures, accessed 12/1/2009).

*Interventions*

**Balloon angioplasty** is an intervention performed during a coronary angiogram. A balloon is inflated inside the narrowed artery, pushing aside any residing plaque, and allowing blood flow to resume. A **stent** is a complex metal mesh which can be inserted into the affected artery after balloon angioplasty. A stent acts as a permanent scaffold, holding the artery open, and reducing the chance that the artery will re-occlude. Traditional stents have been known to collect thrombi (blood clots), and lead to re-narrowing, but the majority of stents presently used release a drug to inhibit re-narrowing until the artery wall grows over the exposed stent http://www.patient.co.uk/showdoc/27000306/ (Patient information resource for Balloon Angioplasty procedures, accessed 12/1/2009).

**Coronary Artery Bypass Graft** is presently a less common procedure. An artery is removed from the patient's leg, and grafted across the occluded coronary artery, bypassing it. The procedure is considerably more invasive than Angioplasty, or Stenting as the patient's chest cavity is opened for the procedure. The points where the new artery is grafted can also cause turbulence in the blood flow, leading to an increased likelihood of atherosclerosis developing in those regions in the future. This procedure is typically performed in situations where stenting is not possible

http://www.patient.co.uk/showdoc/40024492/(Patient information resource for Coronary Artery Bypass Grafting procedures, accessed 12/1/2009).

When any of these procedures that were done, a Cardiologist reported the result of the procedure independently.

## Study Methods

*Participants*

The participants were patients presenting to Christchurch Emergency Department with symptoms suggesting Acute Coronary Syndrome, for whom the decision to either admit or discharge was considered within the Emergency Department. This was an observational study, so beyond the consenting procedure, patients had no input in the study. Similarly as a non-interventional study, the patients' management was not altered from standard protocol in any way by this study.

In order to be recruited in the overall cohort, the Emergency Department physician who saw them had to consider the possibility of ACS. Other inclusion criteria were: Patients had to be 18 years old or over, have presented with chest pain of suspected cardiac origin, and received the standard ACS investigations of an Electrocardiograph and a single TnI blood test during their time in ED. The physician must also have provided a risk estimate with their TnI blood request.

The normal TnI blood test request form ED physicians sent to Canterbury Laboratories was replaced with an alternative form, which required physicians to record the signs and symptoms required by the PREtest consult risk calculator, and provide the clinician's subjective estimate of the probability that the patient has Acute Coronary Syndrome via a linear scale. The ward physician's TnI request form was also replaced, but no further information was requested. Canterbury Health Laboratories provided a list of all replaced forms that they processed, and where the form had been sent from (ED or a ward). This allowed the patients who received serial blood tests to be clearly identified. Patients were

then divided into two subgroups depending on whether they were admitted or discharged. Patients for whom both a Ward request form and an ED request form were registered within a single admission were recruited to the "admission" cohort. Any patient for whom an ED form was registered, and were then discharged from ED was recruited to the discharge cohort.

A criterion for the admission subgroup was that the patient must have received a second TnI blood test, which is the next step in the ACS investigation pathway. Patients who were admitted, but for whom no Ward form was registered were excluded because ACS had either been excluded in ED, and their admission was on the basis of an unrelated condition, or the Ward physician failed to complete the form.

Patients who had ST-Elevation (described below) on their first ECG were also excluded because the risk of ACS is too high for this group for the physician to seriously consider discharging them from the Emergency Department. It is standard management for these patients to be admitted, often going directly to treatment without further testing.

All physicians were working at Christchurch ED during the study. The physicians ranged in experience from Junior House Officer (having just completed Medical School), to Senior Consultants, and Professors of Emergency Medicine (both of whom completed Medical school 10 years ago or more, and may have many further qualifications). Although each request form was signed by a physician, the high turnover of ED staff makes tracking individual physicians prohibitively difficult. During the course of the study, over 200 physicians worked in ED. In order to identify each physician, and who they treated, each of these physicians would need to have provided informed content. If a single physician had declined to consent, it would have resulted in every patient that physician treated also being

excluded. Thus, in the interest of maintaining a high study population, the present study observed risk estimate behaviour of ED physicians as a whole. A potential follow-on study could observe how the result found here varies across numerous staff attributes, such as expertise age and personal values.

*Test Methods*

*Follow-up*

A health-index search, and search in the death registry showed whether the patient had died or re-attended hospital within 45 days of their index hospital visit. If either had occurred, the patient's notes were audited to investigate whether the event was cardiac related. In some cases where the patient had died in the community, the patient's GP or Rest Home was contacted to confirm the cause of death. Cardiac-related events between 45 days and 1 year of the index attendance were considered unrelated, but still noted.

*Diagnostic Definition*

The definition of final outcome used to classify patient final outcome was based on the American Health Association definition of ACS (Pollack et. Al, 2002). There is no gold standard test available for ACS.

Any patient for whom an intervention was deemed necessary was immediately considered ACS positive. For patients that received no intervention, investigations were divided into three categories.

The first category was ECGs, and Blood Tests. If either of these investigations was positive, and no other investigations were performed, the patient was considered ACS positive with one exception: If the ECG abnormality was an ST Elevation, and the Biomarkers were negative, this is indicative of Pericarditis, an unrelated condition and is considered ACS negative.

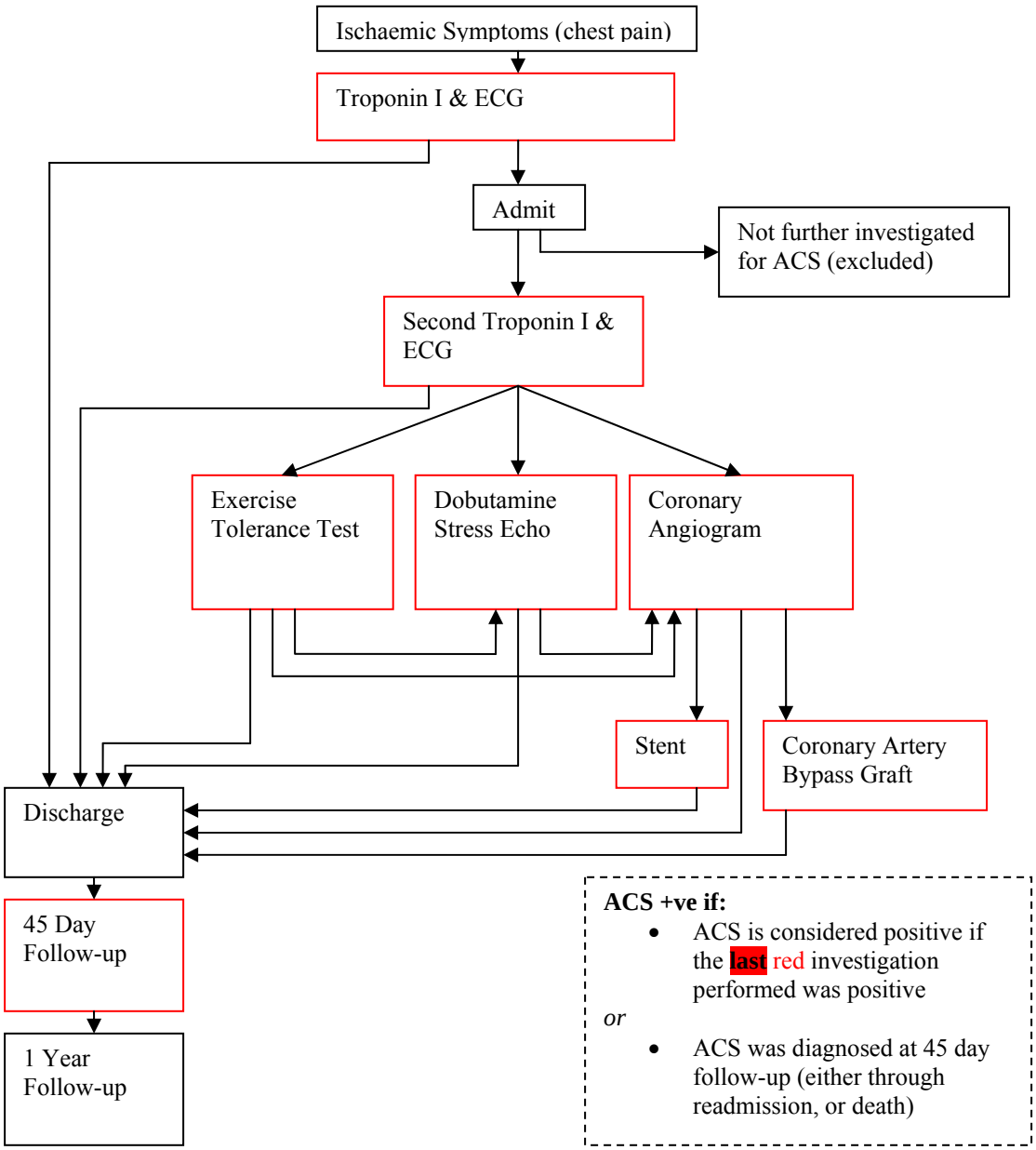
The second investigation category was Exercise Tolerance Test and Dobutamine Stress Echo. A positive result in either of these tests indicates that the heart is not coping when placed under stress, which is highly suggestive of ACS. A negative result indicates that the heart is able to cope under stress, which is not suggestive of ACS. Whether positive or negative, the results of these investigations superseded the results of any first category investigations, unless the Blood test had shown Troponin levels well above what is considered normal. Finally, the third investigation category was Coronary Angiography. This investigation allows the physician to see the coronary arteries directly, and observe whether any of the major vessels are narrowed, or blocked. If performed, the result of this investigation superseded all other investigations.

Any patients who were diagnosed with ACS through the process described above, or died from ACS within 45 days were considered ACS positive. ACS-related re-admissions, and deaths beyond 45 days were noted, but not considered related to the index admission. An ACS-related readmission more than 45 days later is unlikely to be related to the earlier presentation.

The present study was concerned with clinically significant ACS. Any patient who was not found to have ACS upon investigation, and did not have an ACS related death or readmission

within 45 days was not classified as having clinically significant ACS at the time of attendance.

**Figure 2. Diagnostic Pathway**

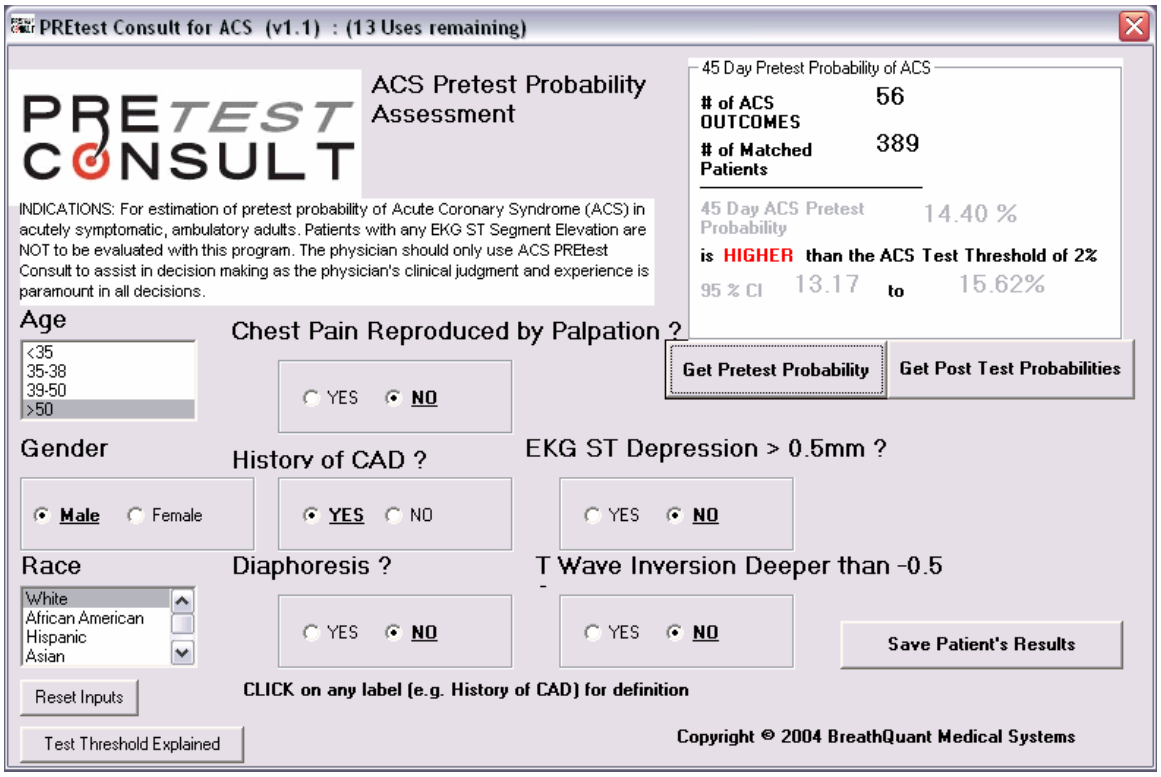


*Physician Risk Estimate*

The Physician's risk estimate was recorded while the patient was still in ED. After the physician's initial examination and the first ECG, the physician ordered a TnI blood test if a potential diagnosis of ACS was being realistically entertained. At this point the physician completed the modified blood request form (appendix 1, form 1) which required the physician to mark on a linear scale from 0 % to 100 %, the probability that this patient would suffer ACS within 45 days, based on the available information. The distance from "0%" to the physician's mark was divided by the overall scale length to provide the risk score as a percentage.

*Risk Calculator*

The PRE-test consult is a data-matching program that was derived from a large multivariate analysis of 150 signs and identified that Age, Gender, Ethnicity, Sweating, Pain Reproduced with Chest Pressure, ST Depression or T-Wave Inversion were the best predictors of ACS. The database came from a large US study (which also excluded patients with ST-Elevation for the same reasons as described above), and has been validated on a US population. Validation on a New Zealand population is currently being performed.




*Note*: Although ethnicity is divided into multiple groups, the calculator presently only differentiates between "white/asian" and "non-white/non-asian".

Before the TnI blood test is ordered, the physician enters the patient's details into the program, which matches the unique constellations of inputs against a 15,000 patient database. The program identifies all patients with the same constellation of results, and what percentage of that group was eventually diagnosed with ACS, to produce a pre-test probability. For each patient, the necessary signs, symptoms, and ECG results were recorded, input into the program, and pre-test probability recorded.

*Post-test probability*

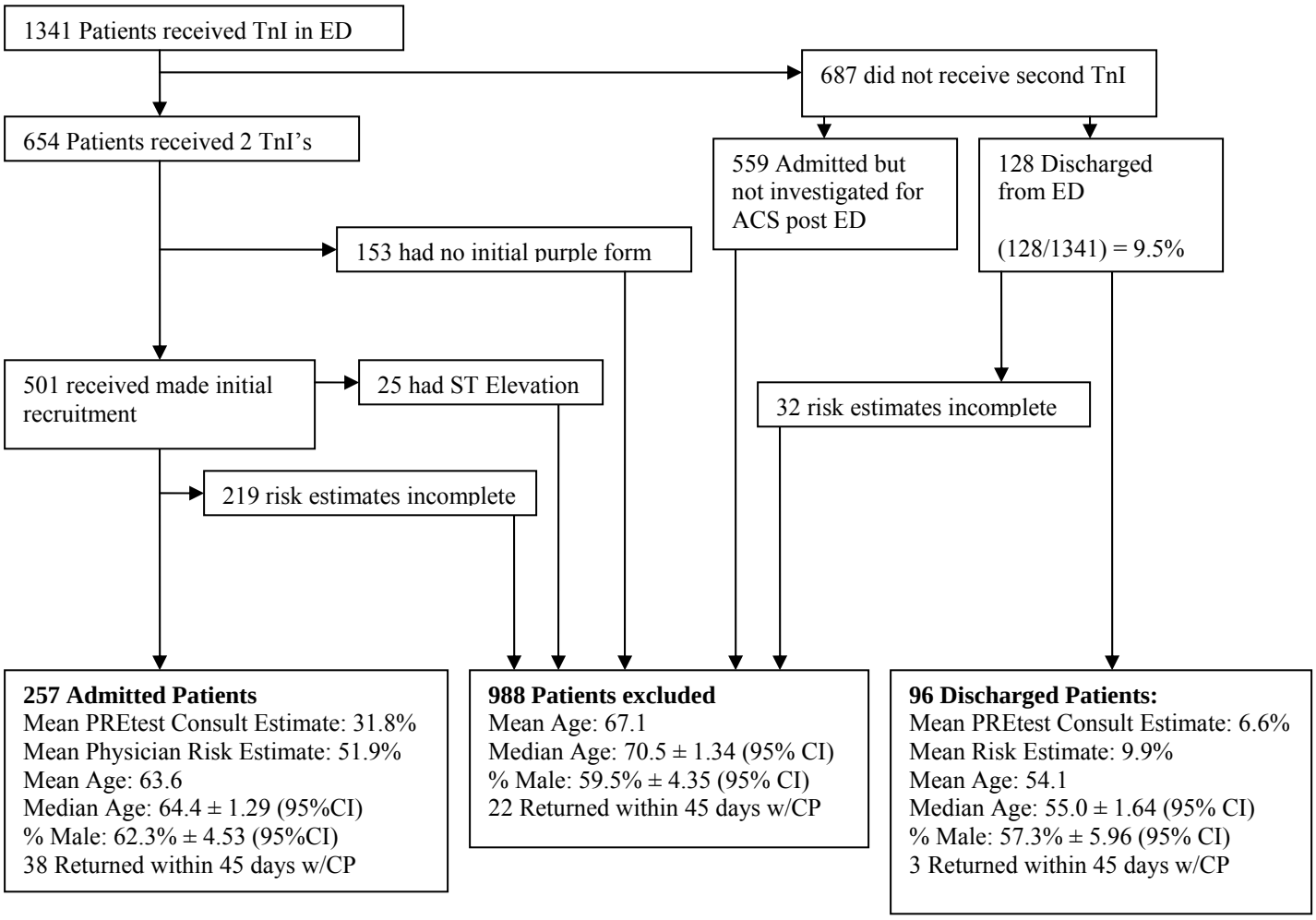
It is important to emphasize that the Pre-test probability was used to decide whether to order the TnI blood test, and not whether admit or discharge a patient. The admit/discharge decision was undertaken after the TnI blood test result was available.

Thus, in order to validate the reported admit/discharge threshold, the pre-test probability must be modified to include the TnI blood test result. As a test with a high sensitivity, and poor specificity, the result of this test greatly influences any prior pre-test probability. The post-test probability for each patient was calculated by multiplying the pre-test probability by the negative predictive value (if the test was negative) or positive predictive value (if the test was positive) of the TnI blood test.

# Results

## Descriptive Statistics

**Figure 3. Recruitment Flowchart**




Of the 353 patients included in this study, 180 (60%) were male. The mean and median age of this group was 60.4 and 61.1 respectively.

## Sub-group analysis

*Admitted/Discharged*

An ANOVA revealed that the observed difference in age between admitted and discharged patients was significant $F(1,351) = 12.9$ ($p<0.001$). The mean physician risk estimate of the admitted sub-group was 52.15%, whereas the mean physician risk estimate of the discharged sub-group was 31.83%. An ANOVA indicated this difference to be significant $F(1,351) = 40.60$ ($p<0.001$). The mean PREtest consult risk estimate of the admitted sub-group was 9.9%, whereas the mean PREtest consult risk estimate of the discharged sub-group was 6.6%. An ANOVA also found this to be significant $F(1,351) = 20.896$ ($p<0.001$).

The fact that both estimation methods yielded significantly different means between the admitted and discharged subgroups indicates that both physicians and the PREtest consult risk calculator were differentiating patients admitted from those discharged. However, the large difference between estimation values suggests that the two estimation methods may not be using the same scale. The interaction between the two estimation methods is explored in detail later.

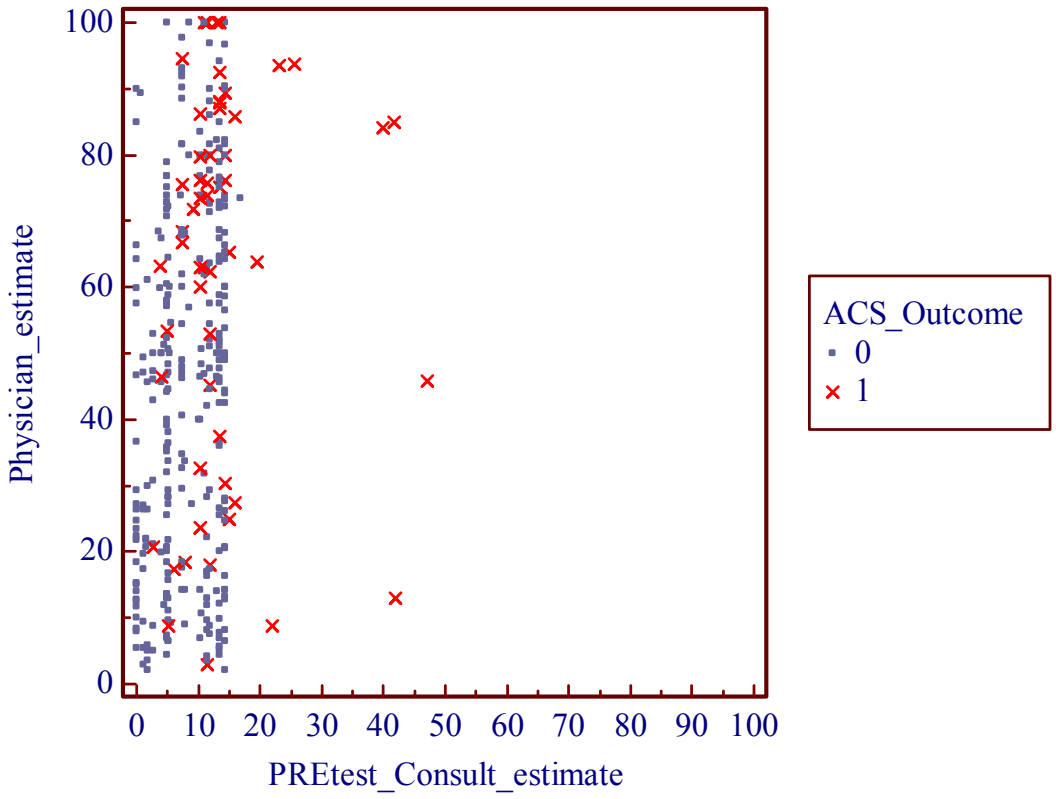
*ACS/Non-ACS*

Fifty-five patients (with a mean and median age of 63.3 and 63 years respectively) were eventually diagnosed as having ACS at the time of presentation. Three of these patients had been discharged, while 52 were admitted. The 3 patients who were discharged were later admitted within 45 days with ACS. Of all patients with ACS, 39 (70.9%) were male. An ANOVA reveals that the observed difference in age between the ACS and Non-ACS group was non-significant $F(1,351) = .394$ ($p=0.531$).

A correlation between the structured risk estimate provided by the PREtest consult risk calculator, and the Physician's estimate can provide some insight into how similar the

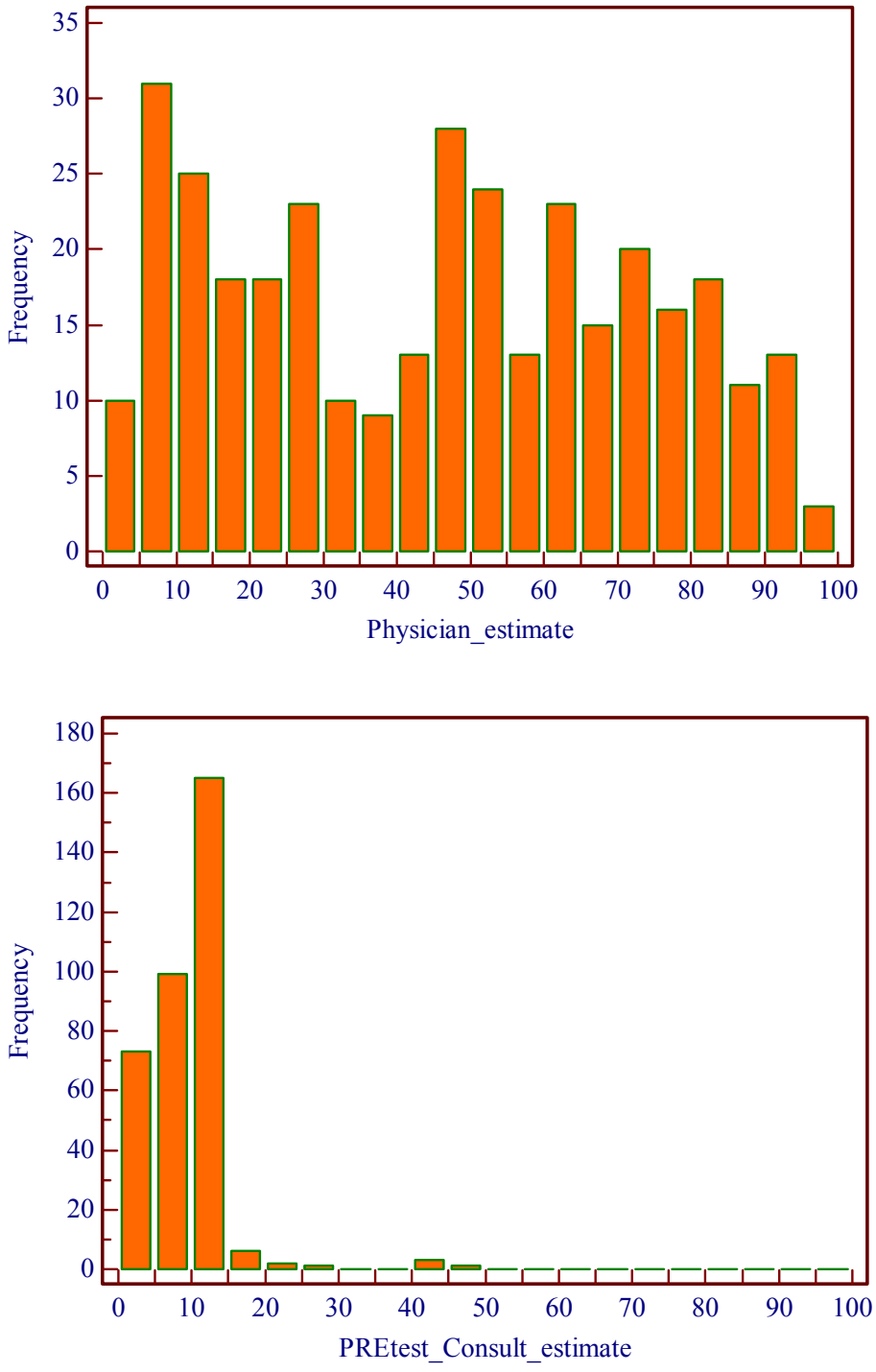
two risk estimates are. A very high correlation ($r \approx 1$) would indicate that physicians use very similar methods to those utilised by the risk calculator. However, a poor correlation ($r \approx 0$) would indicate that physician's estimate digresses from this method in some way. It is important to note that the correlation cannot indicate the cause of the discrepancy.

**Figure 4. Illustration of the correlation between Physician Estimate and PREtest consult estimate**




It is worth noting that the strong ceiling effect observed across the PREtest consult axis is not reciprocated in the Physician axis; across which the data points appear more homogenously distributed. This suggests (at least on a visual level) that the two estimation methods have little in common. The histograms in figure 5 further display the difference in distribution.

**Figure 5. Comparison of PREtest consult and Physician risk estimation distributions**



Physician estimates, and PREtest consult estimates showed a weak, yet significant correlation $r = 0.236$ $p < 0.0001$. With a weak, yet significant correlation, it can be concluded that as suggested visually, the two risk estimation methods share at least some common methods; however, additional factors result in physician estimates significantly digressing from PREtest consult estimates. As the PREtest consult estimates are structured, this poor correlation suggests that physicians may not use an unstructured (and thus potentially uncommunicable) estimation process. This is detailed in the discussion.

It is important to consider that a simple correlation as performed above cannot identify whether the variation is caused by a variation in risk estimation methods from physician to physician, or some common method shared by all physicians, which differs from that of the PREtest consult calculator. In order to measure this, individual physicians would need to be identified. This proved methodologically prohibitive, as was discussed in the methods section.

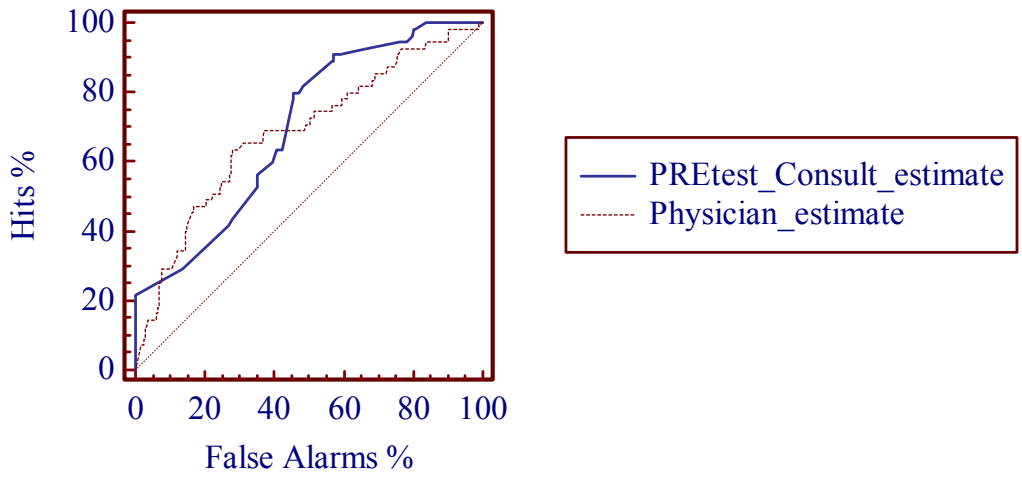
The histograms in figure 5 also suggested a difference in mean risk between estimation methods. To further investigate this, a t-test was performed. The physicians provided a mean estimate of 46.8% with a standard deviation of 28.1, compared to the PREtest consult's mean estimate of 9.04% with a standard deviation of 6.16. The T-test found this large difference to be highly significant ($df = 704$, $t = 24.546$ $p < 0.0001$). At face value, this would suggest that physicians generally feel that ACS is highly common among their patients (and thus vastly over-estimate the prevalence of the disease, with clear implications for the patient-physician communication process). However, a closer analysis of the physician's responses and behaviour is needed before any such

conclusions could be drawn. The conclusion that can be safely drawn at this stage is that physician estimates and PREtest consult estimates differ significantly.

Perhaps more importantly, although the correlation and t-test results identify a difference in risk estimation, neither analysis is able to identify which measurement strategy resulted in better estimations. This distinction holds strong implications for the shared decision-making model. If a structured (and thus communicable) risk estimation process underperforms, then it could not be ethically implemented, as it would expose more patients to the risk of misdiagnosis.

To measure this, ROC curves were calculated for each estimation method. As stated in the methods section, the reference standard for whether any given patient actually had ACS was whether the diagnosis had been made within 45 days via further investigations, interventions, or at post-mortem for those that had died within this time period.

**Figure 6. Comparison of Physician and PREtest consult ROC curves predicting ACS outcome**

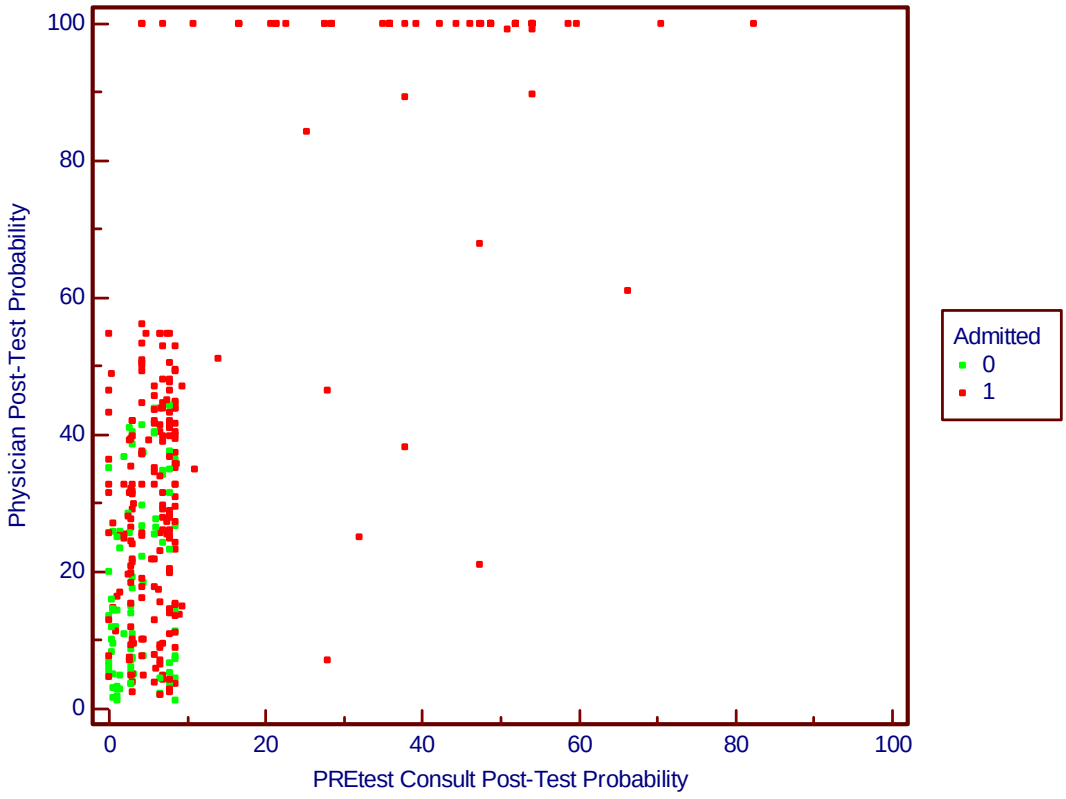



The Physician estimate predicting ACS provided an Area Under the Curve (AUC) of 0.69, whereas PREtest consult provided an AUC of 0.70. The difference was not found to be significant $z = 0.228$ ($p = 0.821$). A number of interesting observations are available from this. The first is that both estimation methods provide comparable accuracy in predicting the patient's condition. As discussed above, this suggests that either method could be used interchangeably without exposing patients to unnecessary risk. However, mitigating this point is the second observation is that the predictive ability of each method is not similar across the entire scale. Physicians appear to outperform the PREtest consult in the intermediate risk estimates, while the PREtest consult appears to outperform physicians in both the upper and lower extremities. So the overall population would not be exposed to additional risk by changing estimation methods, but sub-populations (such as the intermediate risk group) may be exposed to additional risk.

The next step in the analysis is to gain some further insight into how physicians may be calculating their reported risk estimations, and whether these estimates predict physician behaviour. An observation is that the significant difference in age identified between the admitted and discharged subgroups is not reciprocated between the ACS and non-ACS subgroups. Put simply, physicians admitted an older group of patients than they discharged, but the ACS patients were not significantly older than the non-ACS patients. The difference suggests that physicians feel that older patients are more likely to have ACS than younger patients, but that age is not a strong factor in actuality. In the context of shared decision-making, this could result in physicians overstating the risk of disease to their older patients, and understating it to their younger patients.

Before making any further observations of physicians' actual behaviour, it is important to realise that one further investigation is performed before the physician makes their admit/discharge decision. The physician orders the TnI blood test, and in light of the result of this test, makes their decision. Thus, to compare the risk estimate to the physician's behaviour, the result of this test must be taken into account. A negative blood test result significantly reduces the probability that a patient has ACS, while a positive blood test significantly increases the probability. Both risk estimates were modified to account for each patient's blood test result, based on the positive and the negative predictive values of the blood test. So the initial probability estimated would decrease if the blood test was negative, and increase if the test was positive. Henceforth, these modified estimates shall be referred to as the "Post Test Estimates". The post test estimates were also correlated.

**Figure 7. Correlation between Physician Post-blood test Estimate and PREtest consult Post-blood test estimate**

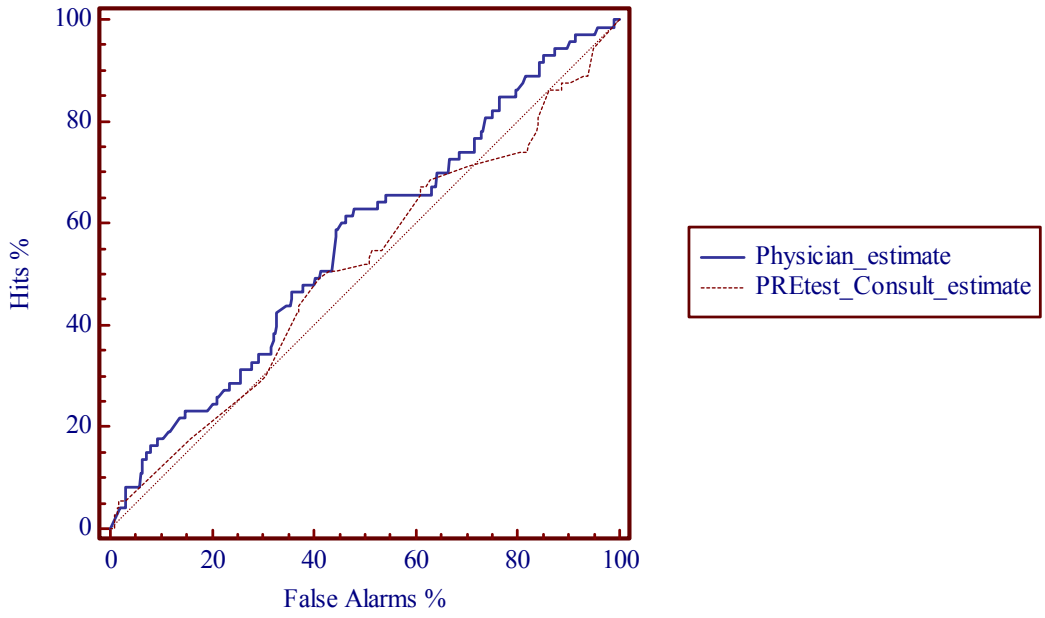



It becomes immediately clear, that the post test estimates have much more in common with the PREtest Consult than their pre-test derivatives, with a relatively strong correlation ($r = 0.784$ $p < 0.0001$). The mean estimates of each estimation method have drawn slightly closer, with the physician mean at 39.7% with a standard deviation of 32%, and the PREtest consult mean at 12.8 %, but a t-test shows that the difference between these means is still highly significant ($df = 704$, $t = 11.269$ $p < 0.0001$). So the estimates are now strongly correlated, but are still not achieving similar values for their estimates.

This provides some insight into how the physician's original estimate may have differed from the structured estimate. This suggests that perhaps physicans should wait until the results of this test are available before engaging in shared decision-making.

The change in correlation in light of this test result raises the question of whether physicians were in some way predicting the blood test result. To test this hypothesis, the ability of each risk estimate to predict the blood test result can be compared via ROC curve analysis.

**Figure 8. Comparison of Physician and PREtest consult ROC curves predicting Blood test result**



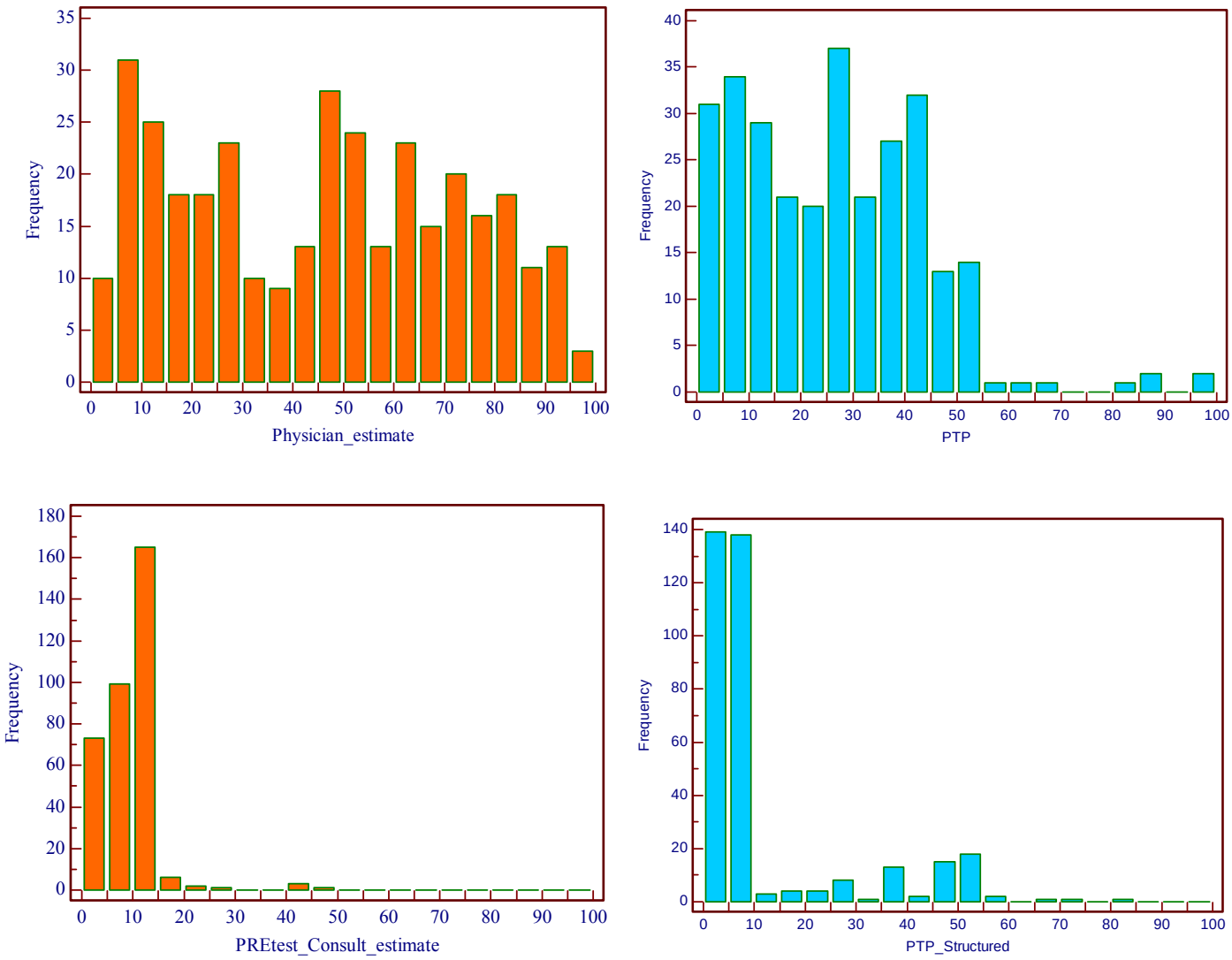

Both risk estimates perform poorly at predicting blood test results. With Physician estimates achieving an AUC of 0.566, and PREtest Consult estimates achieving 0.514, neither estimation method achieved predictive power above chance. The difference is not significant z = 1.12 (p = 0.263). Thus, it can be concluded that the factor causing physician estimates to differ from the structured estimate has no relation to the patient's blood test results. So the inclusion of the blood test result reduces the variance between the estimation methods without either method having a significant relationship to blood test result.

On a side-note, the result of this ROC curve analysis validates that the predictive value of the blood test does not overlap with any other part of the diagnostic process, and is thus a valuable inclusion patient management.

Another potential explanation of the post-test estimates having a stronger correlation is that the blood test result pushes intermediate patient risk estimates on both scales closer to the extremities. This can be seen by comparing the pre-test histograms observed above to post-test histograms.

In order to truly respect this pattern, it must be considered that physicians have consistently been estimating higher values than the PREtest consult risk calculator. Thus, the intermediate risk subgroup resides higher in the physician histograms than on the PREtest consult histograms.

**Figure 9. . Comparison of Pre-test and Post Test risk estimation distributions**

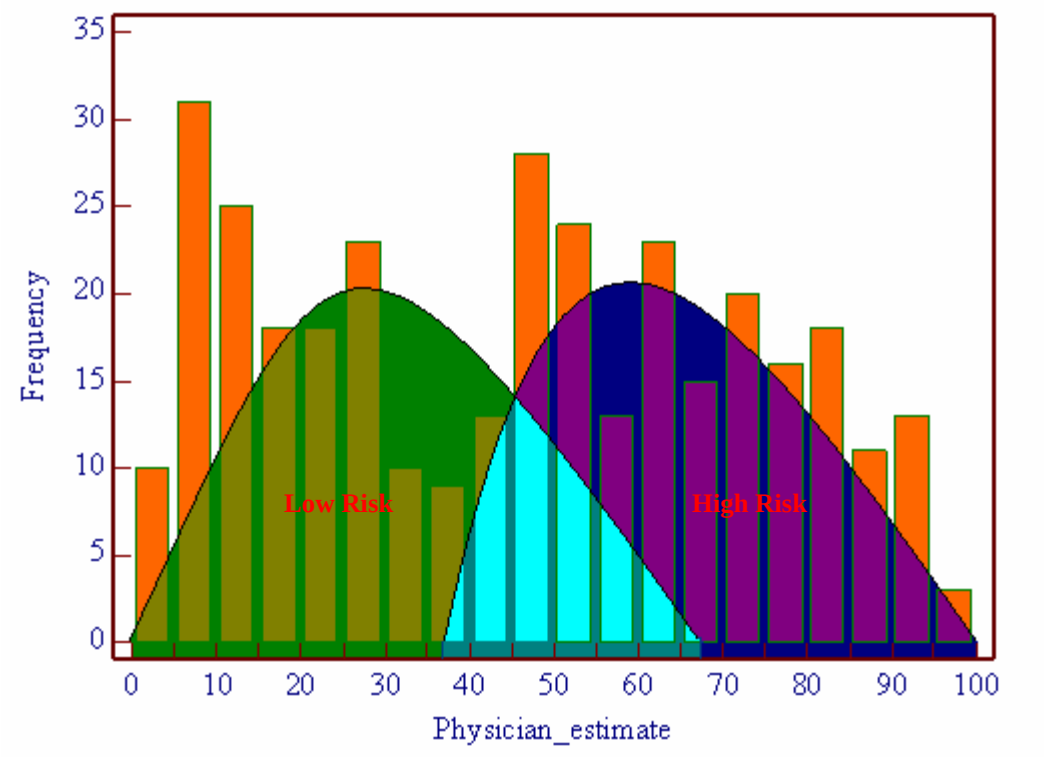



By reducing the intermediate groups, and increasing the high and low groups, the post-test situation is slightly closer to a dichotomous distribution. Theoretically, further testing would continue to erode the intermediate group, and place patients in the low, or high risk groups. A perfect test would isolate all patients without ACS at the absolute lower extremity of the scale (0%), and all patients with ACS at the upper extremity (100%). At this stage, a perfect dichotomy would be achieved.

With this argument in mind, a new hypothesis emerges; It is possible that physicians stratify patients dichotomously rather than quantitatively. To clarify, physicians may be dividing patients into two undifferentiated groups: Low risk and high risk, and manage them accordingly. A surprising test result, such as an unexpected test result, could cause a "low risk" patient to be moved into the "high risk" group or vice versa.

If physicians were performing a dichotomous decision process, then the observed variance in quantitative risk responses may in fact be random. Physicians may simply consider a patient as "low risk" and elect a "low value" on the scale at random. Referring back to the histogram of physician responses, it is possible that two population groups are present.

**Figure 10. Estimation of "Low Risk" and "High Risk" subgroups**



This could somewhat explain how two risk estimates could have little in common, both hold good predictive power, and hold more in common when further stratified. However, another hypothesis that could equally explain this is that physicians differ systematically in their estimations. Perhaps patients given an estimate in the upper distribution were seen by younger, less experienced physicians, fearing a missed diagnosis while the lower distribution was populated by more experienced physicians.

However, this argument could at least be challenged on the grounds that only an abrupt change in physician estimation could explain such a bipartisan distribution. If physician estimation gradually improved with experience, it would be expected that a group of physicians with intermediate experience would have populated the central range of the distribution, which is clearly not the case. However, still further alternatives arise. It is possible that physicians adopt two estimation methods. For example, perhaps physicians are risk averse only when the department is not busy. The only way to ensure that the distribution is not the product of between-physician variation would be to control for such variables as physician experience, and departmental crowding; neither of which were feasible in this study.

# Discussion

## Interpretation of Results

This study aimed to observe whether physicians will be able to communicate the necessary risk information to their patient for shared decision-making to be possible. This study also aimed to compare the testing threshold value obtained in a previous study with the actual behaviour. Finally, this study aimed to measure the reliability of the physician's estimates by comparing physician opinion to a structured risk estimate calculated on an internationally validated risk stratification tool. This was done in order to observe whether physicians would be able to communicate this information to their patient in a shared decision-making model.

In light of this, the significant weak correlation found between Physician and a structured risk calculator such as PREtest consult estimates indicate that physicians share at least some common risk stratification methods with the calculator, but that the physician's gestalt decision process also differs considerably from the structured method of the PREtest consult risk calculator. This coincides well with Gigerenzer's findings, that even people highly experienced in statistical method (such as physicians) are subject to biases and heuristics (Gigerenzer et. al, 1995). The exact nature of this discrepancy may be difficult to ascertain, but the fact that admission and discharge groups differ significantly in age, while ACS and non-ACS groups do not, suggests that one such variation between risk estimate methods is that physicians may consider age as a greater risk factor for ACS than is necessary. As described in the introduction, risk behaviour that appears irrational at face value often has complex rational explanations beneath the surface (Mayo et. al., 1990)..A study into the personal values of physicians may assist in explaining the observed discrepancy.

**Addressing the Testing Threshold Model**

The clearest finding from this study was that physician risk estimates were considerably higher than either the PRETest or their own diagnostic behaviour. This strongly challenges the accuracy of the physician's reported estimates of 2%.

If physicians truly considered 2% as a low enough probability to consistently cease further investigation (as is assumed by the testing threshold model), it would be expected that all patients receiving a blood test would either have a pre-test estimate greater than 2%, or that a positive blood test would result in a post-test estimate above this level. However, patients with pre-test estimates as low as 0.12% were further investigated, and even admitted.

Similarly, cardiac investigations should have ceased in all patients with a post-test estimate below 2%, and all patients with a post-test estimate above 2% should have been admitted for further investigation. In contrast, some patients with a post-test estimate nearing 0% were admitted, while others were discharged. Patients were also discharged with post-test estimates ranging as high as 51.9%.

Thirdly, many patients had a pre-test estimate so high that even a negative blood test would not have reduced their risk below the 2% testing threshold, theoretically making the investigation superfluous. Physicians are strongly encouraged to only order a test if the result will influence the way that patient is managed. Like the patients presenting with ST-Elevation, patients with such a high pre-test probability should have theoretically bypassed the troponin blood test, and proceeded directly to more invasive investigations

if a completely structured decision process was being followed. In contrast, many of these patients received a negative blood test result and were then admitted; indicating that the blood test result was not effectively used in the physician's decision. Two possible explanations of these discrepancies occur; either the physicians are not utilising their own quantitative estimates in their decision process, or the physicians are looking for evidence that confirms their own judgement rather than for evidence that might challenge it.

Finally, it was frequently observed that two patients with identical presenting signs and symptoms were managed differently. Assuming the physician's decision process is rational, there are only two possible explanations for this: The first is that the same variables are evaluated differently by different physicians (based on the physician's expertise, or values), or that the physicians are consistent with one another, but measuring at least one variable not accounted for in this data set. Although not immediately evident, the implications for shared decision-making are the same for both explanations.

If the first explanation is true, and the diagnostic decision process varies between physicians (regardless of whether it is by personal values, or personal experience) then the physician's risk assessment cannot be structured, and thus cannot be communicable. It must be stressed that the physician's role in a shared decision-making process would be to provide reliable risk data to the patient. If the data is being reliably assessed communicated, then two physicians should provide the same information for the same patient. Internal variation in risk assessment indicates that the physicians are subjectively augmenting the risk data before providing it to the patient.

If the second explanation is true, and the physicians are consistently measuring at least one additional variable, then the physicians must be applying at least some emotional or value-based consideration to their risk factor selection and evaluation. To clarify, it must be considered that the PREtest consult risk calculator was created by measuring over 150 variables in a database of 15,000 patients (far more cases than any individual physician could have seen in their career), and selecting the 8 variables with the greatest predictive value3. If the physicians are selecting additional variables, it is against practical evidence, and must be subjective. For example, physicians may be placing emphasis on the severity of the pain, even though pain severity has been shown to have little predictive value for ACS.

It is worth mentioning the third alternative; that physicians are performing a more complex analysis than the risk calculator. This is not listed above, as it has been excluded as an unfeasible explanation for the observation. The argument is that the physician may be simply including the $9^{th}$ most significant risk factor in their analysis, or accounting for some high-order interaction between certain risk factors (for example, perhaps severity of pain is significant if the patient is over 50). The reason that this can excluded from the list of potential explanations is because the physician assessment did not outperform the risk calculator in predicting ACS, although admittedly it did not significantly underperform either.

Through standard statistical methods, the inclusion of further variables will reduce variance, and increase predictive power, but sacrifice the degree of freedom in the data (Walker et. al. 1940). Variables will only be included if the reduced variance justifies the reduction of freedom. Thus, if the physicians are including further relevant factors in

their risk assessment, it would be expected that they would outperform the calculator. Similarly, if physicians were including a superfluous variable in their estimation, then it would be expected that this would actually introduce more variance, reducing the predictive accuracy of their estimates. Neither of these is observed.

The final argument that could be proposed is that physicians are performing a structured analysis, with a number of variables included above those in the PREtest consult, but also with some variables omitted. This could result in a significant variation between estimation methods, without a significant sacrifice in predictive power. Although this third option cannot be logically discredited like the two above, it does fall into the realm of reasonable doubt. It is simply highly unlikely that two different structured estimations would result in such similar performances.

**Physicians' Decision Process as a Rational Dichotomy**

All of these observations strongly suggest that the physician's decision process is at most, only semi-structured, and may incorporate additional factors beyond those accounted for by the PREtest consult risk calculator that were not measured in this study. These may include the physicians' personal values, experience, or perhaps statistically insignificant (but highly emotive) presenting symptoms.

However, it must also be considered that of the 96 patients that were eventually discharged from ED without investigation, only 3 had adverse outcomes (none of whom died). This suggests that the overall risk of the discharge group was approximately 3%. While above the level physicians provided as their testing threshold, this figure is considerably closer than the physician estimates suggest. Thus, while the physicians risk

estimations may appear erratic, particularly when compared to those obtained from a completely structured estimation; the physicians' actual decision process appears to be relatively safe and effective.

One potential explanation of the variation between the physicians' risk estimates and physicians' behaviour is that physicians are not performing quantitative risk estimation, but are rather focusing on a dichotomous outcome. Perhaps physicians accurately come to the conclusion that a patient is "safe for discharge", with little interest in the exact risk of disease. This may also explain why physician estimates were typically higher than PREtest consult estimates.

It is possible that physicians were simply nominating a high figure at random for patients for whom they were considering admission, and figures generally considered low for patients whom they were considering discharge. To clarify, perhaps physicians would elect a probability of 80% for patients with an actual risk of 20% as the difference between a probability of 20%, and a probability of 80% are inconsequential in terms of how that patient would be managed. They were simply indicating that the patient should be admitted. Similarly, the physician may simply elect a risk of 5% for a patient they are planning to discharge because 5% is generally considered a "low" figure, albeit not considered acceptably low for this context.

To further support the theory that physicians perform dichotomous risk management, a decision aid was developed for an unrelated condition called Pulmonary Embolism. The decision aid (known as the "Geneva Score") collected a series of important risk factors and symptoms, and then divided patients into 3 groups: Low, Moderate, and High risk

accordingly (Wicki et. al. 2001). Physicians supported the principle of dividing patients into groups based on statistical evidence, but were dissatisfied with the "Moderate" risk group. There was concern over how management of these patients should differ from the low and high risk groups. Subsequently, the "Simplified Geneva Score" was developed. The same risk factors were collected, but patients were divided into only 2 groups: "Low risk" and "High risk" (Klok et. al. 2008). Physicians found this dichotomy highly useful.

It is important to stress that the original Geneva Score had been statistically derived, and held a high predictive power. Thus it was only incongruence between the decision process of the tool and the physician that led to its modification. The Modified score was not favoured for its predictive power, but for its ease of use. It was simply closer to the physician's preferred decision processes.

**Potential Implications**

From the perspective of a solitary decision-maker, a dichotomous decision process is reasonable, and potentially desirable, given that this situation provides only two management strategies, and a dichotomous outcome. However, a lack of structured or quantitative risk estimation holds important implications for the prospect of developing a shared decision-making process between physician and patient. As mentioned in the introduction, one of the principle assumptions of the shared decision-making model is that the physician will be able to effectively collect and communicate the necessary information to the patient for them to be able to make an informed decision, based on their personal values.

The diagnostic process described in the methodology is complex (as detailed in Figure 2.) and the final decision is made after following complex branches of reasoning with numerous tests and procedures providing novel information at different stages. It is reasonable to expect that for

patients to provide a constructive contribution to the decision-making process, that they would need to have at least some global understanding of this diagnostic process before they provide their input. Given that patients do not currently hold such an intimate knowledge of diagnostic process, it would become the physician's responsibility to communicate this to the patient during their consultation.

A failure at any stage of this communication process would result in the patient-physician relationship reverting back to one of paternalism. If the physician's current risk estimation process is not structured, or quantitative, it would be expected that at present, they will have difficulty in communicate their own estimates effectively. Furthermore, if their current decision-process is in fact dichotomous, the physician will struggle to inform the patient without influencing their decision.

Thus, the primary conclusion of this study is that physicians are currently not in a position to implement a shared decision-making relationship with their patients without some form of training. At present it is expected that physicians will struggle to convey the necessary information to their patient, and any communication that does occur may be unintentionally misleading. Given that physicians' risk responses in this study falsely suggest that the physician decision process is irrational, it is likely that patients would similarly misinterpret their physician's responses.

Alternatively, it could be argued that physicians and patients operate under the same biases; and that effective communication would be preserved, even in the absence of accurate risk estimates. This alternative results in the same apprehension. Before implementation, it would need to be objectively confirmed that communication is actually preserved. Even if physicians and patients share the same biases, it is possible

that the "double handling" of the risk information could result in these biases becoming inflamed.

Effectively, the decision to implement shared decision-making is a risk based decision in itself. Although the potential benefits are great, the risk of a shared decision model causing a detrimental influence in patient care is simply too great at this stage to encourage implementation.

**Future Research**

Beyond investigating the "double handling" issue raised above, a key question this study raises is whether the physician's existing decision process can be changed safely. In order to answer this question, further research needs to investigate which formats of communication patients would comprehend. Gigerenzer et. Al. (1995) suggest that natural frequency formats are a naturally intuitive method for delivering quantitative information to patients, but given the rich variety of formats available, many more studies will need to be completed before a confident conclusion could be reached as to which format is most effective.

Another assumption being made is that all patients will respond equally to a single format. This assumption also must be tested. At present it cannot be excluded that one group of patients might respond well to a list of outcomes, and their statistics, while another group prefers the each option to be summarised in terms of overall life expectancy. Belavkin (2001) suggests that a patient's problem solving abilities are dependent on their present mood. This would mean that even a single patient could potentially prefer one format on one occasion, and another the next. If this is found to be

the case, and there is no single format that is universally understood by patients, this will further complicate the shared decision-making process, as physicians would need to learn how to identify which format to use with each patient during their consultation, and adapt their decision process accordingly.

Assuming a single format is found, the nature of the physician's current decision process then needs to be better understood. The main question of interest here is whether the physician's decision process can be safely modified to become more amenable to the format patients prefer without reducing the overall effectiveness of the physicians' decision process. The major concern is that the physician's existing decision process may have developed because it is the best way for them to make sound medical decisions. If training them to use a different (more communicable) decision process results in them making more errors, then the entire process will have become self-defeating. Elwyn et. Al. (1999) measured physician responses to the proposed shared decision-making model. Although most physicians felt they were currently not trained in the skills required to engage in shared decision-making (supported by physician's responses in this study) the main finding was a very wide range of suggestions as to what role the physician would play in a shared decision process, from hotly defending paternalism, to giving patients free reign on their own health care. Such poor consensus among professionals as to what is best for the patient should emphasise the necessity for a cautious and evidence-based transition; for the risk of endorsing a sub-optimal decision-process is high.

An alternative solution is to train physicians in translating their existing decision process into any communicable format required, so that their existing decision-making is not affected, but they are also capable of discussing it with any of their patients. This is

perhaps a more feasible option, as it requires less interference with the physician's established (and generally effective) methods. This results in less new information for the physician, and less risk of having a detrimental influence on the physicians' existing (and generally effective) decision process. However, for this solution to be seriously considered, the assumption that a single format will be universally comprehensible to patients still needs to be validated, and accommodated. It also needs to be investigated whether physicians are capable of learning how to convert their decisions into any given communicable format.

For shared decision-making to be considered effective and practical, both the physician and the patient must feel confident in the information that has been gathered, and confident that the other party has fully understood what they have said. For example, if it is found that physicians do not feel confident in performing a quantitative decision, then they will revert to a method they feel comfortable with, regardless of whether it is communicable or not. Similarly, if the physician suspects the patient has not understood the information they have provided, they may feel compelled to speak on the patient's behalf. Unless both stages of this process are successful, the patient-physician relationship will always involve at least some degree of paternalism.

**Conclusions**

In summary, it appears that a physician's risk estimate, although weakly related to structured risk stratification, cannot be effectively compared to a structured, quantitative risk analysis. As such, it is likely that physicians would currently have considerable difficulty converting their existing risk estimation process into a communicable format. This may pose a serious challenge to the prospect of effective shared decision-making.

The primary questions remaining are whether physicians can adopt a novel decision-process, or learn how to communicate their existing one to patients. Overall, many hurdles still lie in the path of achieving effective shared decision-making. However, the arguments in favour of adopting a shared decision-process compel investigators to persist in contributing to the current understanding of the existing decision process, identifying the current impediments to a shared decision-process, and to continue to build solutions to those impediments.

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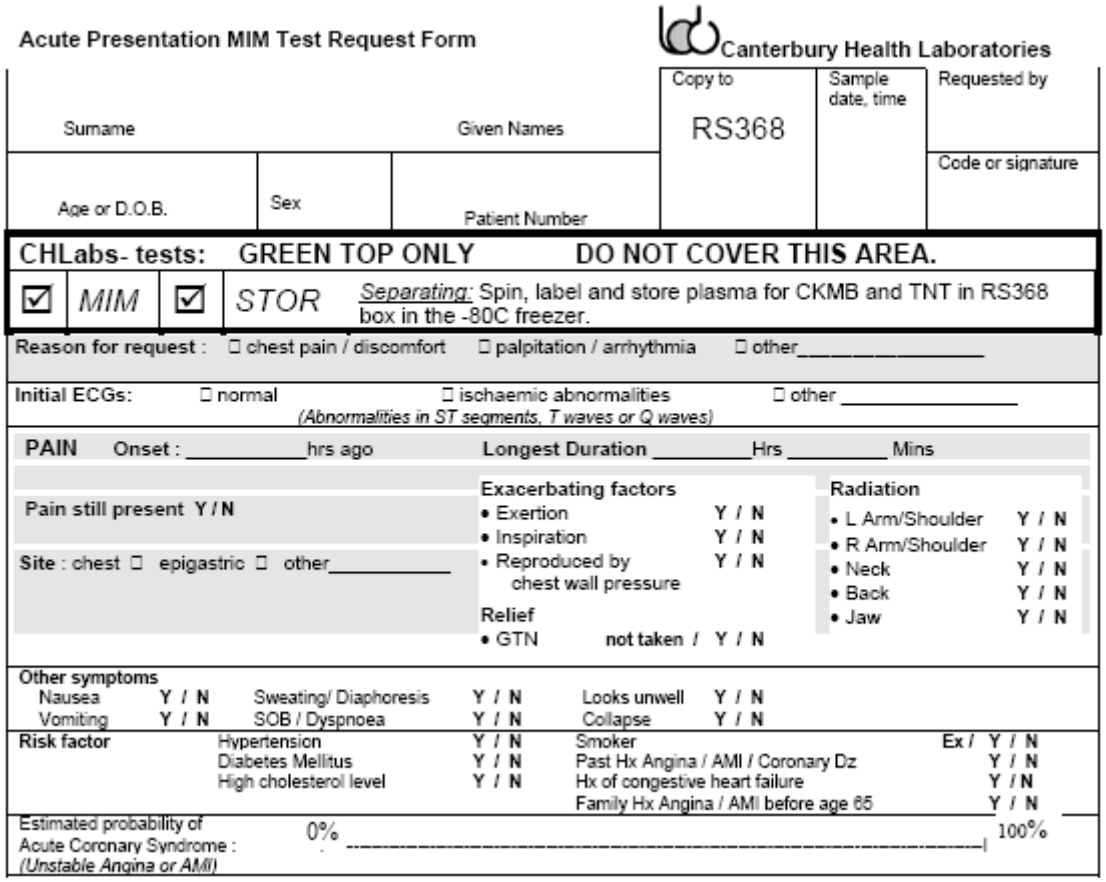
# Appendix

## Form 1



Acute Presentation MIM Test Request Form

Canterbury Health Laboratories

| Surname | Given Names | Copy to RS368 | Sample date, time | Requested by |
|---|---|---|---|---|
| Age or D.O.B. | Sex / Patient Number | | | Code or signature |

**CHLabs- tests: GREEN TOP ONLY DO NOT COVER THIS AREA.**

☑ MIM ☑ STOR *Separating:* Spin, label and store plasma for CKMB and TNT in RS368 box in the -80C freezer.

**Reason for request :** ☐ chest pain / discomfort ☐ palpitation / arrhythmia ☐ other_____

**Initial ECGs:** ☐ normal ☐ ischaemic abnormalities ☐ other _____
*(Abnormalities in ST segments, T waves or Q waves)*

**PAIN** Onset : _____hrs ago **Longest Duration** _____Hrs _____ Mins

**Pain still present Y / N**

**Site :** chest ☐ epigastric ☐ other_____

**Exacerbating factors**
- Exertion Y / N
- Inspiration Y / N
- Reproduced by chest wall pressure Y / N

**Relief**
- GTN not taken / Y / N

**Radiation**
- L Arm/Shoulder Y / N
- R Arm/Shoulder Y / N
- Neck Y / N
- Back Y / N
- Jaw Y / N

**Other symptoms**

| | | | | | |
|---|---|---|---|---|---|
| Nausea | Y / N | Sweating/ Diaphoresis | Y / N | Looks unwell | Y / N |
| Vomiting | Y / N | SOB / Dyspnoea | Y / N | Collapse | Y / N |

**Risk factor**

| | | | |
|---|---|---|---|
| Hypertension | Y / N | Smoker | Ex / Y / N |
| Diabetes Mellitus | Y / N | Past Hx Angina / AMI / Coronary Dz | Y / N |
| High cholesterol level | Y / N | Hx of congestive heart failure | Y / N |
| | | Family Hx Angina / AMI before age 65 | Y / N |

Estimated probability of Acute Coronary Syndrome : *(Unstable Angina or AMI)* 0% ---------- 100%

## Form 2

# Canterbury Health Laboratories
## RESEARCH REQUEST FORM

| Surname | Given Names | | Copy to | Sample date, time | Requested by |
|---|---|---|---|---|---|
| | | | RS368 | | Martin Than<br>Ext 80270 |
| Age or D.O.B. | Sex | Patient Number | | | |
| Project Code (Care of) | Location | | | Collected by | Beep number |

**NOTE: This sample must be taken >12 hours but <24 hours POST symptoms**

TESTS REQUIRED

☑ *TNI*

☑ *STOR* Separating: Spin, label and store **two aliquots** of plasma for CKMB and TNT in RS368 box in the -80C freezer.

Sample requirements: **GREEN TOP ONLY (Lithium heparin)**

| | | |
|---|---|---|
| **RS368**<br>**Form 2** | **Acute Presentation MIM Study** | Send Form & Specimen to:<br>Canterbury Health Laboratories<br>Cnr Hagley Ave & Tuam Street<br>CHRISTCHURCH |